DISSERTATION ON

"EFFICACY OF PARTICLE REPOSITIONING MANOEUVRE IN BENIGN PAROXYSMAL POSITIONAL VERTIGO"

Submitted in partial fulfillment of the requirements for

M.S. DEGREE BRANCH-IV OTORHINOLARYNGOLOGY

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UPGRADED INSTITUTE OF OTORHINOLARYNGOLOGY

MADRAS MEDICAL COLLEGE

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CERTIFICATE

This is to certify that this dissertation entitled **"EFFICACY OF PARTICLE REPOSITIONING MANOEUVRE IN BENIGN PAROXYSMAL POSITIONAL VERTIGO"** is submitted by **Dr. S.SARAVANAKUMAR,** appearing for M.S. ENT. Branch IV Degree examination in March 2010 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, Tamilnadu, India.

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CONTENTS **PAGE NO:**

INTRODUCTION

There is nothing more gratifying for a physician than managing a disorder that is for the most part easily diagnosed, and more important, simply and effectively treated using noninvasive means. Benign paroxysmal positional vertigo (**BPPV**), the most common affliction of the vestibular labyrinth, is one such disorder.[10]

Benign Paroxysmal Positional Vertigo is one of the common causes of vertigo of labyrinthine origin. It is a peripheral labyrinthine disease and seldom a manifestation of a CNS disorder. It is commonly associated with head trauma, inner ear disease such as Meniere's Disease or vestibular neuronitis. In some cases **BPPV** is idiopathic. The pathophysiologic finding in **BPPV** is calcium carbonate (***otoconia***), deposits on the cupula of the posterior semicircular canal (***cupulolithiasis***) or free-floating otoconia (***canalolithiasis/canalithiasis***) within the endolymph. Vestibular nerve and ganglion neuronal degeneration have been reported in patients with BPPV. It is now accepted that BPPV is caused by canalithiasis and cupulolithiasis of any of the three ***semicircular canals***(**SCCs**), with the posterior canal being most commonly involved.

Generally BPPV is a self-limiting disorder that can resolve spontaneously due to the ability of the endolymph to dissolve otoconia. Traditionally, fatiguing

vestibular rehabilitation exercises were given to patients with persistent BPPV. The introduction of the Canalith Repositioning procedure (CRP) or Particle Repositioning Manoeuvre (PRM) by Semont and Epley has revolutionized the management of BPPV to the point that most physicians, paramedical health care providers and even self-treated patients have used these manoeuvre. Our study is conducted to prove the efficacy of particle repositioning manoeuvres in the treatment of BPPV.

AIMS OF THE STUDY

The present study is under taken

- To evaluvate the efficacy of Particle Repositioning Manoeuvre while treating single canal Benign Paroxysmal Positional Vertigo of any of the three semicircular canals.
- To diagnose exact canal of Benign Paroxysmal Positional Vertigo
- To identify the demography of single canal Benign Paroxysmal Positional Vertigo in adults.

REVIEW OF LITERATURE

HISTORY

1897 Adler described BPPV[23]

1921 Barany is credited with first description of BPPV as syndrome[10,]

1952 Dix and Hallpike described positional test , coined the term BPPV [10]

1969 Schuknecht theory of cupulolithiasis [4,5,10,23](coined the term)[23]

1980 Brandt & Daroff repeatedly provoked symptoms through head positioning exercise by dispersion of otolithic debri from the cupula[24]

1985 Toupet & Semont singler treatment approach[23]

1987 Norre & Beckers Brisk method 52% within one neck

1988 Semont Freyss&Vitte liberatory or Semont maneuver[13]

1991 Parnes , McClure –demonstrated particles in post SCC [18,23]

1992 Epley- theory of canalithiasis[11, 20]

1996 Serefini,Palmieri& Simoncelli[18,19]

50%-one treatment

90%-within five treatment sessions

ANATOMY OF THE VESTIBULAR SYSTEM

The anatomy and physiology of the vestibular system are rather complicated and involve multisystem and neural substrates. For the purpose of description, the system is divided into its peripheral and central components.

The ***peripheral vestibular system*** is the pars inferior of the membranous labyrinth and is composed of the semicircular canals, the otolith organ (saccule and utricle), and the peripheral vestibular nerve. The semicircular canals are three mutually perpendicular canals in the horizontal, vertical anterior, and vertical posterior planes. Each canal on one side lies in the same plane and works conjointly with its corresponding canal on the other side of the head. Thus both end-organs form a 3D gyroscopic arrangement to respond to head motion in all directions, The three canals join the utricle (membranous tube) and saccule at the vestibule in the center of the membranous labyrinth(FIG1). Each canal enlarges at the vestibule, forming the ampulla that houses the crista, the cupula, and the vestibular hair cells. The crista lies inferiorly and forms the base of the cupula and contains the bodies and supporting cells of the vestibular hair cells (FIG3,4A). The cupula extends from the crista to form a tight seal at the superior aspect of the ampulla and separates the endolymph on both sides of the cupula, This anatomy allows the cupula to deflect relative to endolymph movement and subsequent

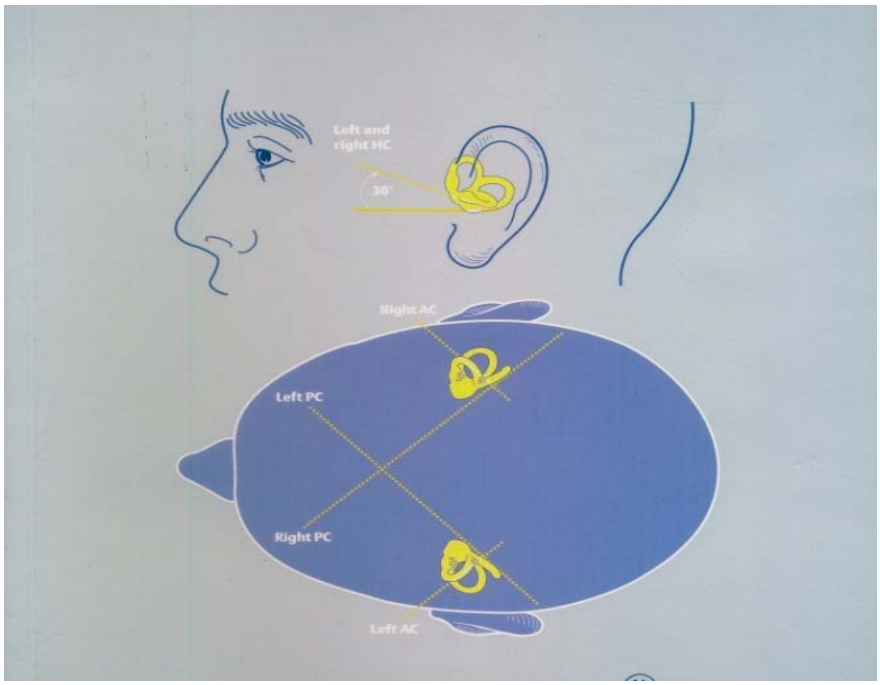

Left and
right HC
30°
Right AC
Left PC
Right PC
Left AC

FIG-1

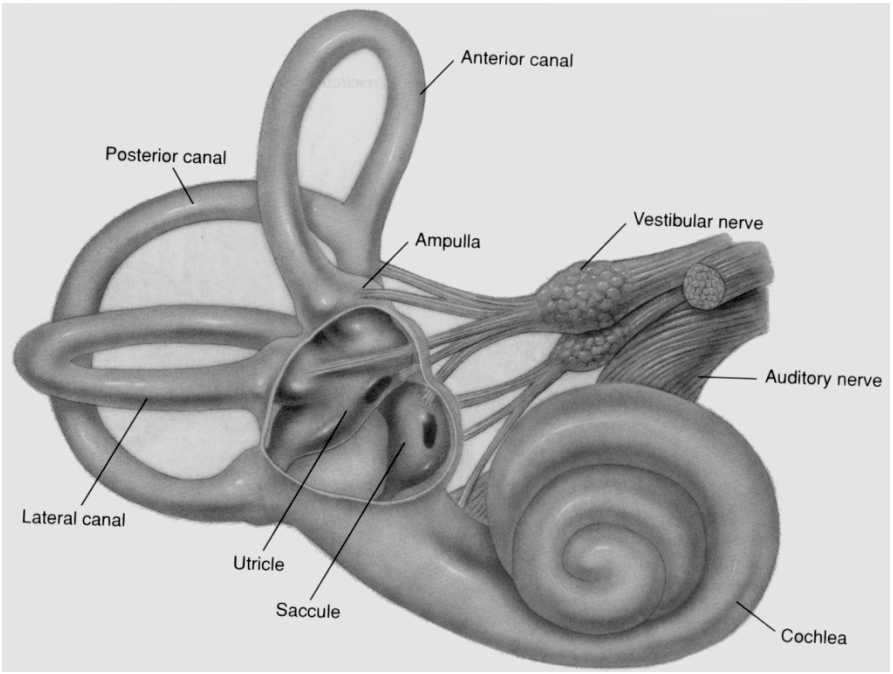

Anterior canal
Posterior canal
Ampulla
Vestibular nerve
Auditory nerve
Lateral canal
Utricle
Saccule
Cochlea

FIG2

excitation or inhibition of the vestibular hair cells.

The utricle and saccule, collectively called the otolith organ, are in the vestibule and oriented approximately in the horizontal and vertical planes. Each organ is composed of the basement membrane (macula) with cell bodies and otoconial membrane in which the hairs extend(FIG3). The utricle plays a large role in postural Control and primarily senses changes in orientation to gravity and horizontal movements. The saccule is similar in organizational structure but it is oriented in the vertical plane.

The semicircular canals are responsible for *detecting angular head movement* in the ***pitch*** (shaking your head "yes") plane, the ***yaw*** (shaking your head "no") plane, and the ***roll*** (tilting your head to the side) plane .The arrangement of the semicircular canals at right angles to each other causes the inner ear fluid(endolymph)to flow toward or away from the ampulla in atleast one canal on each side with any of the above- mentioned head movements. As long as response registered in each of the matched canals on either side of the head is the same, balance and orientation are maintained.

The cell body of vestibular sensory epithelia, **type I** and **type II** are embedded within the crista of the semicircular canals and the basement membrane of the otolith. Type I hair cells fire irregularly with slow kinetics, whereas type II fires regularly and have fast kinetics. Each cell has a single layer and thick hair ,

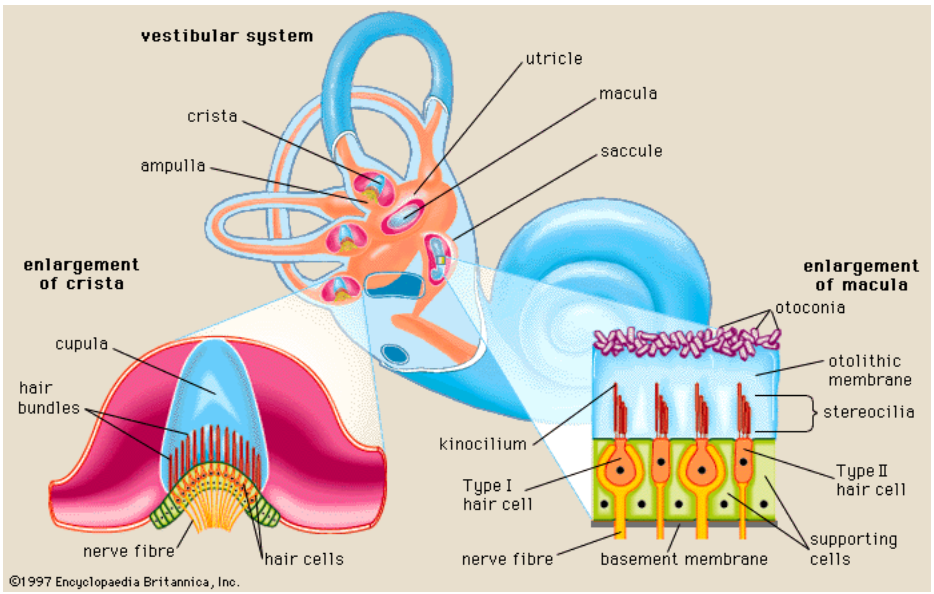


FIG-3

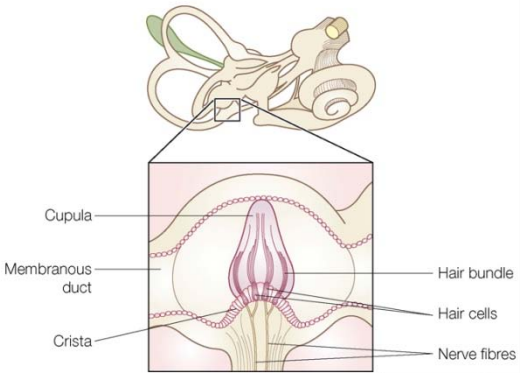


FIG-4A

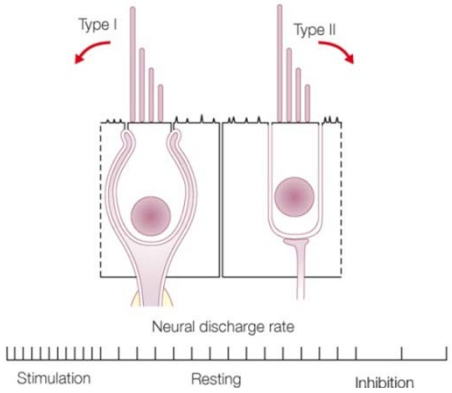


FIG4B

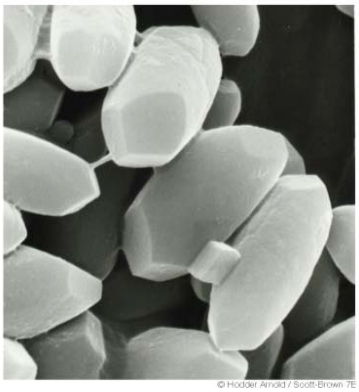


FIG 5A

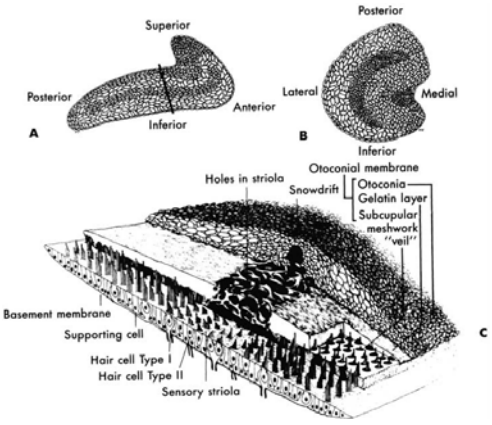


FIG 5B

the ***kinocilium***, and several thin and shorter hairs, the ***stereocilia***. These cells are mechanical to neural transducers that collectively have a spontaneous, resting, firing rate of approximately 100 spikes per second.

Movements of *stereocilia toward the kinocilium excite*, that is **depolarize,** the cells and lead to increases in their resting firing rate, where as movements *away from the kinocillium inhibit,* that is, **hyperpolarize**,, the cells and lead to a decrease in their resting firing rate(FIG4B). The kinocillium and stereocillia orientation determines the excitation and inhibition pattern of the semicircular canals. The kinocillium is oriented towards the vestibule in the horizontal canals and away from the vestibule in the vertical canals. In the otolith, the sensory hairs are arranged in multidirections on both surfaces with the kinocillium located towards the stereocilia in the utricle and away from it in the saccule. This orientation ensures excitation of the canal or the otolith in the direction of head motion and inhibition of the ones in the opposite direction. For example, with right head angular motion, the right horizontal canal is excited and the corresponding posterior canal is inhibited. This excitation-inhibition activity is the essence of vestibular physiology.

The vestibular nerve is part of cranial nerve VIII and is formed by the superior and inferior division. The superior division innervates the vestibular ganglia of the horizontal canal, the anterior canal ,the utricle, and part of the saccule. The

inferior division innervates the vestibular ganglia of the posterior canal and the saccule(FIG2) .The resting rate is modulated by the stimulation and inhibition of the nerve to maximum of 400s/s and a minimum of zero.

"Nystagmus" refers to the repeated and rhythmic oscillation of the eyes. Stimulation of the semicircular canals most commonly causes "***jerk nystagmus***," which is characterized by a slow phase (slow movement in one direction) followed by a fast phase (rapid return to the original position). The nystagmus is named after the direction of the fast phase. Nystagmus can be horizontal, vertical, oblique, rotatory or any combination thereof. "***Geotropic nystagmus***" refers to nystagmus beating towards the ground, whereas " ***Ageotropic nystagmus***" refers to nystagmus beating away from the ground.

THE CENTRAL VESTIBULAR SYSTEM

The key components of the central vestibular system are the ***vestibular nuclei complex*** in the brainstem. These nuclei lie in the floor of the fourth ventricle in the pontine area bound laterally by the restiform body , ventrally by the nucleus and spinal tract of the trigeminal nerve, medially by the pontine reticulate formation and abducent nucleus. A schematic diagram of the central vestibular system is shown (FIG 6A).

The vestibular nuclei are divided in to four subgroups: the *lateral, medial,superior*

and inferior nuclei. They receive primary afferent signals from the vestibular nerve in addition to multisensory afferents from the contralateral vestibular system, reticular formation, cerebellum, spinal cord, and neck muscular and bony structures. The efferent signals include the lateral vestibulospinal tract, the medial vestibulospinal tract, the vestibuloculomotor tract, the otolith ocular tract, the vestibulospino cerebellar tract, and the vestibulothalamic-cortical tract.

The major tracts are the vestibulospinal, the vestibuloculomotor, and their connection with the cerebellum, These tracts control posture and head movements during active and passive motion.

PHYSIOLOGY

The main function of the vestibular system is to generate informatiom for the central nervous system with a four-fold purpose. (FIG 6B)

1. Provide general orientation of the body with respect to gravity
2. Enable balanced locomotion and body position:
3. Readjust autonomic functions after body reorientation and
4. Ensure gaze stabilization

The vestibular portion of the inner ear is anatomically suited for two main functions ***stabilization of gaze*** during rapid impulsive head movements and ***postural control*** in a gravitational field. To function optimally, labyrinthine input

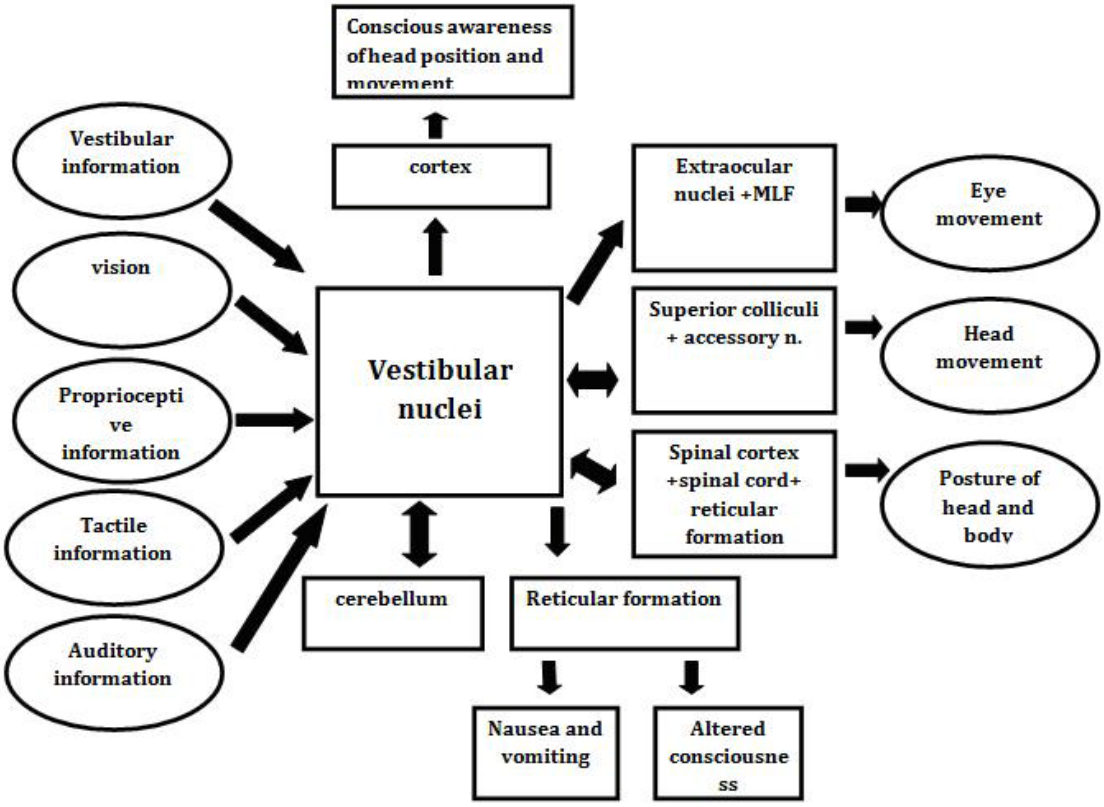
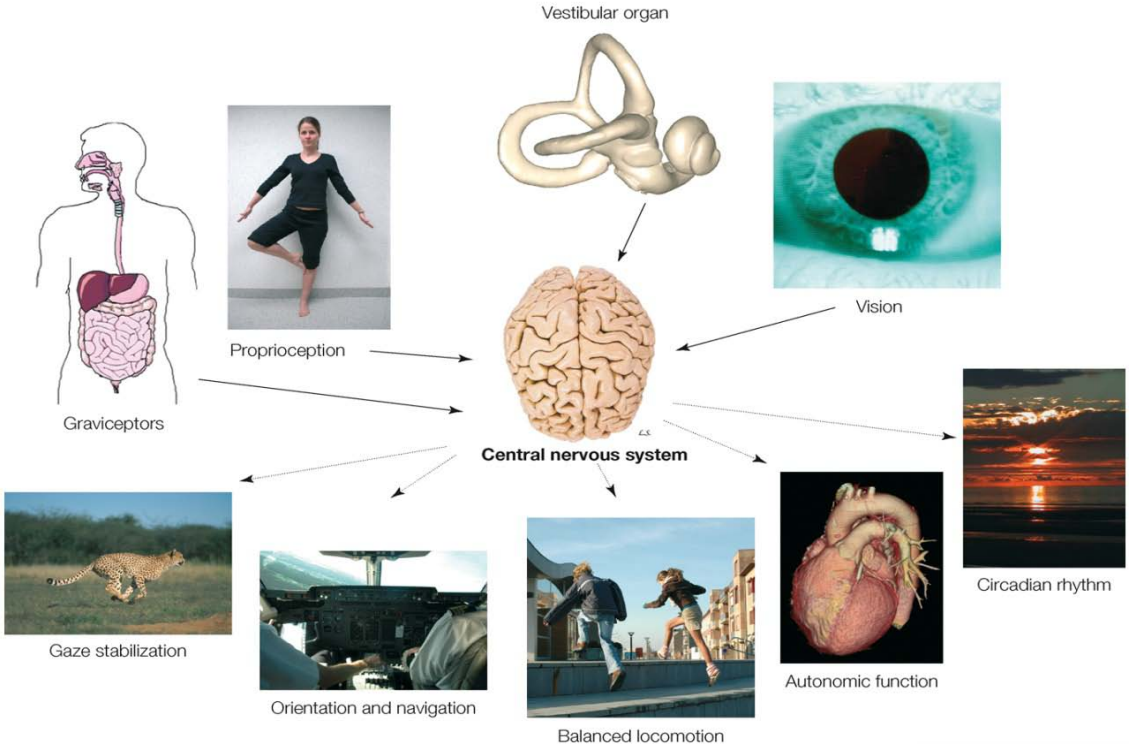

Conscious awareness of head position and movement
cortex
Vestibular information
vision
Propriocepti ve information
Tactile information
Auditory information
Vestibular nuclei
cerebellum
Reticular formation
Nausea and vomiting
Altered consciousne ss
Extraocular nuclei +MLF
Superior colliculi + accessory n.
Spinal cortex +spinal cord+ reticular formation
Eye movement
Head movement
Posture of head and body

FIG-6A


Vestibular organ
Vision
Proprioception
Graviceptors
Central nervous system
Circadian rhythm
Gaze stabilization
Orientation and navigation
Balanced locomotion
Autonomic function

is combined with visual, proprioceptive, auditory, and other sensory cues within the brainstem so that resultant eye and body movements are in context with the task at hand.

Angular head acceleration causes relative endolymph flow in a direction opposite to that of the head movement, which displaces the cupula. cupular displacement in turn causes bending of the hair cell bundle, resulting in a change in membrane potential. due to hair cell orientation on the cristae of the horizontal canals, ampullopetal flow(towards the ampulla) causes hair cell depolarization , whereas ampullofugal flow (away from the ampulla) causes hyperpolarization.

In the posterior and superior canals, the hair cells have an *opposite orientation*, and this pattern of excitation and inhibition is reversed.

Otolith hair cell cilia are embedded in the gelatin layer of the otolithic membrane of the utricle and saccule that underlies the layer of otoconia FIG 5A & B (composed primarily of calcium carbonate crystals).because the otoconia have a higher specific gravity than endolymph, their greater inertia causes them to be displaced upon the hair cell layer by applied linear acceleration (including gravity) in the plane of the otolithic membrane

The organ of ***sensory transduction*** in the otoliths consists of the maculae of the utricle and saccule. The hair cells of both otolith organs are oriented at right

angles to a central curvilinear axis called the ***striola***. Oppositely oriented hair cells are separated by the striola, giving the maculae bidirectional sensitivity.The utricular macula is oriented roughly perpendicular to the earth-vertical plane in normal head position(i.e ,roughly in the same plane as the horizontal semicircular canal), and the saccular macula roughly parallel to the earth-vertical plane.

Two reflexes play main role in maintenance of equilibrium. They are

1) ***The vestibuloocular reflex (VOR)*** to ensure gaze stabilization

2)***The vestibulospinal reflex (VSR)*** to ensure maintenance of an upright position of the body and trunk and head stabilization in space

The purpose of the VOR is to stabilize images on the retina particularly during rapid impulsive head movements. These movements can be highly variable in frequency and velocity, such as voluntary changes in head position, subtle perturbations

. Earlier theories described a simple "***push – pull" arrangement*** in which a head movement would cause a excitatory response in one vestibular nucleus and corresponding inhibitory response in the contralateral vestibular nucleus .Now it is believed that both inhibitory and excitatory response occurs in both vestibular nuclei for each head movement. Inhibitory responses are the result of *inhibitory commissural reactions* from the contralateral vestibular nucleus these commissural connections help to explain the process of tonic balancing.

The **horizontal rotational component of the VOR** is easiest to assess. Head rotation in the earth-horizontal plane primarily involves activation of the horizontal semicircular canals and minimal activation of other canals. Hair cells in the crista of the canal towards which the nose rotates are depolarized, whereas those in the contralateral side are hyperpolarized. Ipsilateral afferent firing rates are modulated upward from their resting rate, whereas contralateral afferent firing rates are modulated downward.

The anterior (superior) and posterior canals participate primarily in the **vertical VOR** in response to rotation in the pitch plane(around the interaural axis).Bilateral anterior canal stimulation causes upward eye deviation. Bilateral posterior canal stimulation causes downward eye deviation .Unilateral stimulation of any of these canals results in an unopposed torsional component in addition to those mentioned. This torsional component is always such that the upper poles of the globes deviate away from any individual canal. This results in torsional nystagmus with quick phases directed toward the ipsilateral ear, with upbeat quick phases for posterior canal stimulation and downbeat quick phases for anterior canal activation.

Interaction between canal and otolith functions is important for compensating head movements while viewing near targets. When viewing targets at visual infinity (only a few meters in actual practice), the amount of eye

movement required to refoveate an image following a linear head movement is negligible. For near targets, however, the compensatory eye movement required is a function of the vergence angle of the eyes and the relative position of the target with respect to the individual visual axis of each eye. This eye movement can be measured and the parameters of the linear VOR determined. Another otolith-ocular reaction is ocular counter-rolling, which may be seen as a torsional rotation of both eyes in response to a sustained lateral head tilt in the opposite direction(FIG6C)

Vestibulospinal reflex:

VSR is concerned with *postural adjustments*. postural adjustments can be either volitional or reflexive.VSR is associated with reflex postural control. there are four reflexive postural control strategies:

1. ***Ankle strategy***- contraction of leg muscles to keep the body aligned with the hips & knees, effective for small, slow perturbation.

2. ***Hip strategy***- movement of head and hips in opposite directions to counteract weight shift from centre of gravity.

3.***suspensory strategy***- increases the stability by lowering centre of gravity.

4.***stepping strategy***- when other attempts to maintain centre of gravity fails, it establishes a new centre of gravity by a stepping motion.

The main function of the vestibular system is to maintain accurate spatial

orientation during activities of daily living .

The vestibular system is also critical to survival and safety. The functional task are stabilization of gaze while the head is in motion, to maintain clear vision; stabilization of body position; balance, to avoid falling and to correct for posture at the limits of stability; and adaptation to new balance tasks and compensation. This achieved through **VOR, VSR, and cerebellum** to provide appropriate and optimal motor command for intended task. This process is highly complex and dependent on the fact that the vestibular system is fastest acting sensory system, much faster than the visual and proprioceptive systems. In fact, in the eye movement system cannot respond instantaneously to the vestibular signals that are stored for delayed acquisitions by the eye movement system, a process commonly referred to as "**velocity storage of VOR"**. The exact neuroanatomic substrate for this storage system is unclear, but it is believed to be in the brain stem reticular formation and modulated by the vestibulocerebellum. Autonomic function adjustments occur after alterations of body orientation.

BENIGN PAROXYSMAL POSITIONAL VERTIGO

Clinical features

Benign paroxysmal positional vertigo(BPPV) is by far the most common cause of positional vertigo. Moreover BPPV is the number one vestibular disorder, causing about 25% of referrals to specialized dizziness clinics .

Benign : not a very serious or progressive condition

Paroxysmal : sudden and unpredictable in onset

Positional : comes with a change in head position

Vertigo : causing a sense of dizziness

The prevalence of BPPV increases with advancing age, women being affected almost twice as often as men. BPPV may involve each semicircular canal, with BPPV of the posterior canal being the most common variant. Episodes may be associated with first noticed in bed, when waking from sleep.

Patients often describe the occurrence of vertigo with lying down or getting up ,getting in and out of bed, Rolling over in bed, Bending over picking something up ,Looking up or down (top-shelf vertigo),Shaving, Washing hair in shower, Going to

dentist or beauty salon. Any turn of the head bring on dizziness associated with nausea and vomiting. There is no new hearing loss or tinnitus

Predisposing factors :

Inactivity

Acute alcoholism

Major surgery

CNS disease

Causes:

Idiopathic

Trauma

Ear disease

Otitis Media

Vestibular neuritis

Meniere's disease

Otosclerosis

Sudden SNHL

TYPES:

POSTERIOR SCC BPPV

HORIZONTAL SCC BPPV

SUPERIOR SCC BPPV

BILATERAL BPPV

All subtypes of BPPV can be diagnosed on the basis of clinical observation .The revelation of its pathophysiology has made BPPV the most successfully treatable cause of vertigo.

Classical post SCC – geotropic rotatory nystagmus

Horizontal SCC – purely horizontal nystagmus

Non-fatiguing nystagmus – cupulolithiasis > canalithiasis

Duration – limited < 20 seconds, Reversal upon return upright position

Response decline upon repetitive provocation

Benign paroxysmal positional vertigo of the posterior canal (**PC-BPPV**) causes brief attacks of vertigo which are precipitated by changes of head position. Patients typically experience vertigo when they turn over in bed, lie down from sitting , sit up from lying , extend the neck to look up, or bend over. The illusion of the movement is usually rotator, but a sensation of body tilt can also occur. Other complaints during the attack include imbalance, oscillopsia, nausea and sometimes

vomiting. Patients are usually aware that certain head movements precipitate attacks of vertigo. They often develop strategies to avoid vertiginous attacks, such as sleeping propped up or holding their neck stiff, which may lead to immobility and prolongation of the natural course of the disease. A secondary anxiety disorder with or without dizziness may persist even after BPPV has resolved.

A single attack of **PC-BPPV** usually lasts 5 to 20 seconds and hardly any longer than half a minute. However, after a flurry of attacks, patients may complain of prolonged dizziness and imbalance lasting from hours to days. Typically, PC-BPPV manifests itself with symptomatic episodes lasting from a few days to several months, which are interspersed by asymptomatic intervals of several months to year's duration. Most cases of BPPV are idiopathic, but about 25% develop after head trauma or on the background of pre-existing labyrinthine disorder such as vestibular neuritis or meniere's disease. Bilateral involvement which is rare in idiopathic **BPPV** appears to be more common in post traumatic patients. The diagnosis is confirmed by provocation of vertigo by positional testing and observation of typical nystagmus. The classic test for provocation and conformation of PC-BPPV is a Dix –Hallpike manoeuvre.

Dix- Hall pike positioning for identification of canalithiasis of the left posterior canal.

The manoeuvre starts with the patient sitting upright on an examination couch with his head turned 45 degrees towards the examiner. She is instructed to keep her eyes open and to watch the examiners forehead. Then the patient is swiftly moved to a lateral head hanging postion. The appearance of a transient, torsional upward nystagmus indicates PC-BPPV of the lower most ear. When the patient is upright, otoconia is located at the base of the cupula. (FIG8)

During the dix-hallpike test, the head is rotated backwards in the plane of the posterior canal, inducing movements of the particles within the canal which leads to an endolymph and cupula displacement in the same direction. The ensuing activation of the canals hair cells results in a mixed torsional upward nystagmus which reflects the excitatory connections of the posterior canal with the superior oblique and the inferior rectus muscle. The nystagmus subsides after the particles have reached the most dependent point of the canal and the cupula has returned to the resting position. When the patient sits up again the particles will be shifted in the opposite direction causing inhibitory deflection to the cupula and hair cell , resulting in the reversed nystagmus.

Alternatively, a lateral tilt of the trunk and head from a sitting position can be performed with the head turned 45% to the opposite side, which positions the

head with the lateral aspect of the occiput on to the couch. With both manoeuvres, the actual positioning of the posterior semicircular canal is identical.

The diagnosis of PC-BPPV can be reliably made when the positional nystagmus fulfills the following criteria:

- ***Torsional – vertical nystagmus***: this appears when the patient is positioned to the symptomatic side . The most prominent direction of nystagmus is a torsional component, beating with the upper pole of the eyes towards the undermost ear. In addition, there is a smaller vertical-upbeating nystagmus component, most prominent on the uppermost eye. As nystagmus direction is influenced by direction of gaze, the patient should fixate on the doctor's nose to keep the eyes closed to primary gaze.
- ***Latency***: typically, nystagmus and vertigo start a few seconds after the precipitating head position is reached. Nystagmus intensity increases rapidly and then decays (crescendo- decrescendo)
- ***Duration***: nystagmus usually lasts 5 to 20 seconds and only rarely exceeds 30 seconds.
- ***Reversal*** : a few seconds after the patient has returned to the sitting position, a transient nystagmus of lesser intensity beating in the opposite direction can be observed.

- ***Fatiguability***: vertigo and nystagmus decrease with repeated positioning in most cases. However repeated testing is distressing and is usually not required.

Unilateral PC-BPPV may mimic bilateral BPPV when during positional testing of the unaffected ear .The head is rotated more than 45degrees to the side which stimulates the posterior canal of the opposite (symptomatic)

Pathophysiology

Cupulolithiasis- -otoconia in the utricle break loose and adhere to the cupula of the posterior semicircular canal (FIG7A,B)

BPPV is caused by ***dislodged otoconia*** *from the utricle,* which may aggregate to a clot. When this mass has entered a semicircular canal it causes inadequate endolymph flow after changes of head position. This concept has been termed canalolithiasis. This concept can explain all clinical features of PC-BPPV . Latency is probably caused by movement of the particles within the ampulla (before reaching the narrow duct) where they have only minor hydrodynamic effects. The limited duration of the nystagmus reflects the journey of the particles along the duct before they settle in its lower most part. Nystagmus reversal after sitting up is explained by the reversed direction of particle migration leading to a cupular deflection in the opposite direction .

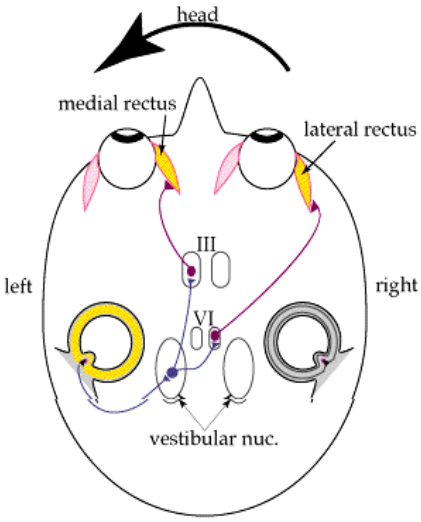


FIG-6C

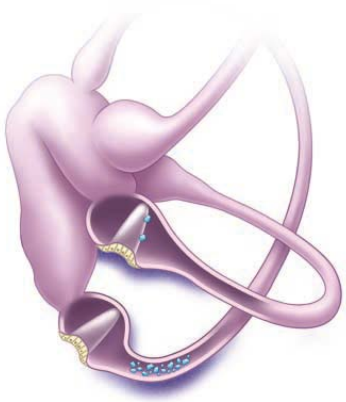


FIG 7A

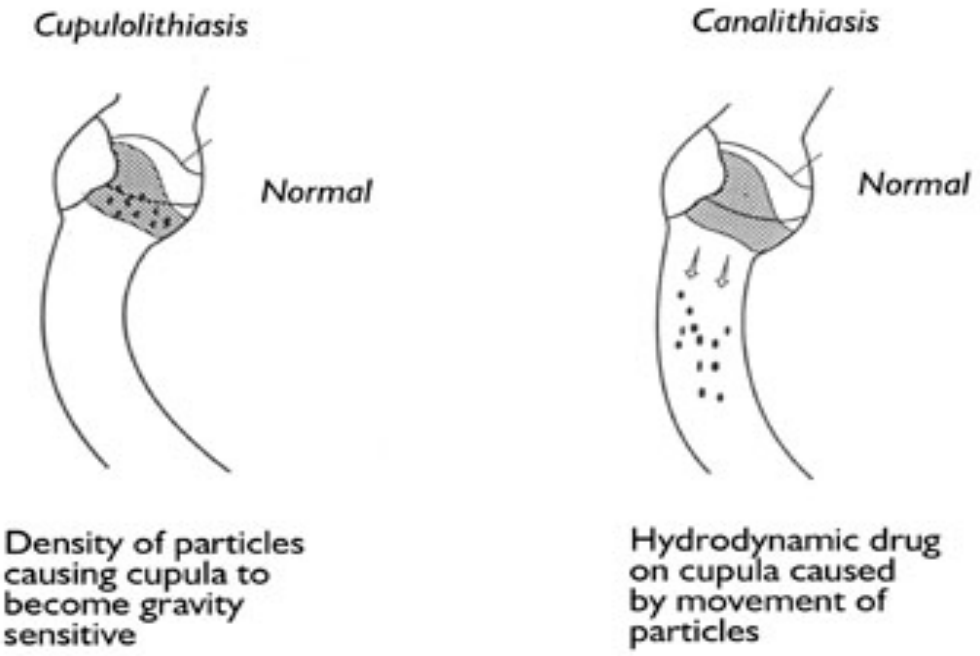


FIG7B

Otoliths (calcium carbonate particles) are normally attached to a membrane inside the utricle and saccule .The utricle is connected to the semicircular ducts. These otoliths may become displaced from the utricle to enter the posterior semicircular duct since this is the most dependent of the 3 ducts Changing head position relative to gravity causes the free otoliths to gravitate longitudinally through the canal. The concurrent flow of endolymph stimulates the hair cells of the affected semicircular canal, causing vertigo

The ***canalolithiasis*** concept is supported by several histological and intraoperative findings. Shchuknecht described granular depostits both on the cupula and within the semicircular canal in patients whose suffered from PC – BPPV prior to death from unrelated disease[12]. He suggested ***cupulolithasis*** or ***"heavy cupula"*** Mobile endolymph particles have been observed intraoperatively within the posterior canal in patients with BPPV. Recovered particles have proved morphologically consistent with degenerative otoconia when studied by electron microscopy. However the most convincing proof for canalithiasis comes from the efficacy of positioning manoeuvres, which clear the affected canal of the mobile particles.

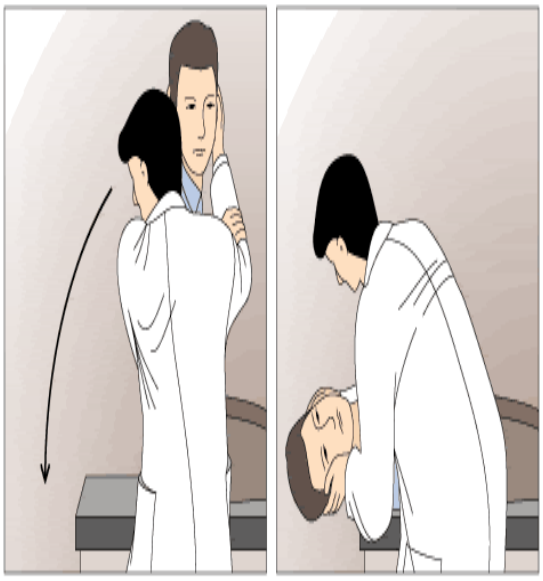
CANALITH JAM:

A third proposed mechanism is thought to be caused by particles creating a blockage in the canal or by particles becoming wedged between the cupula and the adjacent ampulla wall. The resultant nystagmus and vertigo are persistent and unaffected by the patient's head position[10]. This could be managed by combination of the application of vibration with repositioning manoeuvre.[1]

EPLEY'S CANALITH REPOSITION PROCEDURE(CRP) for the treatment OF PC-BPPV on the right:(FIG11)

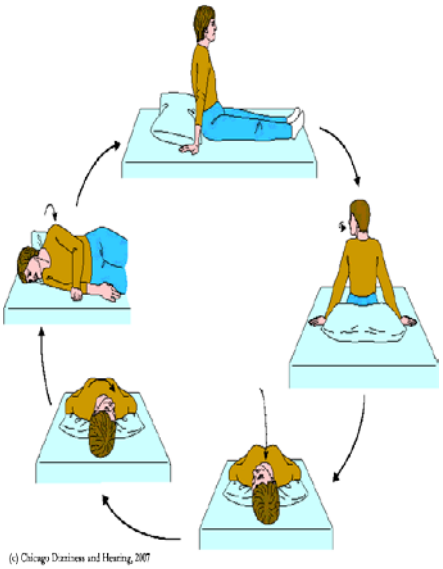
The procedure consists of set of five successive head postioioning that are hand guided by the therapist. Each positioning is performed rapidly and is maintained for 30 seconds. From right to left:

- Sit the patient upright with head turned to 45 degrees to the affected side.
- Lie patient down with head dependent as if performing dix-hallpike manoeurve.
- Rotate head through 90^0 to opposite side with face upwards, maintaining dependent position.
- Rotate head and body further so that the patient is facing obliquely downwards , with nose 45 degrees below horizontal.
- Raise patient to the sitting position.

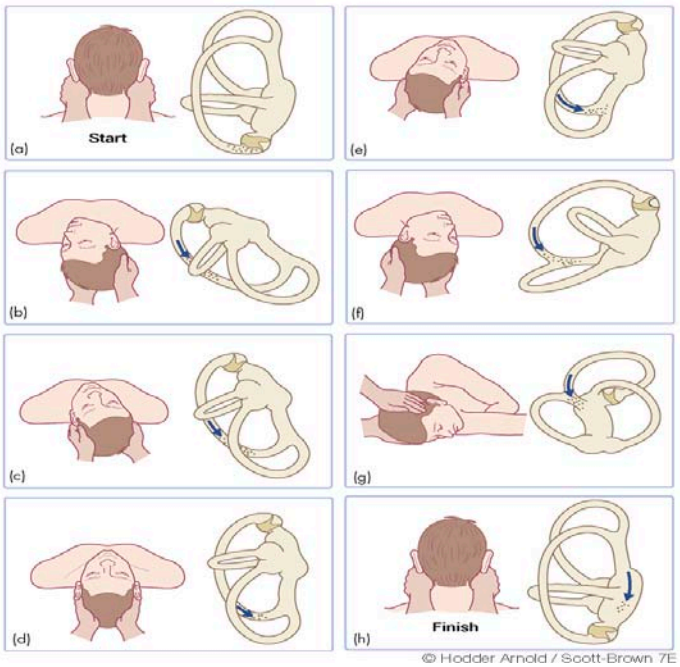


DIX HALLPIKE'S TEST(FIG 8)

SEMONT'S MANOEUVRE(FIG 9)



MODIFIED EPLEY'S MANOEUVRE(FIG 10)



EPLEY'S MANOEUVRE (FIG 11)

SEMONT'S MANOEUVRE ***for the treatment of PC-BPPV on the left***(FIG9):

This procedure is hand guided by the therapist. All movements are performed rapidly. The head is turned 45 degrees away from the affected ear throughout the manoeuvre.

- Sit the patient upright with head turned 45^0 away from the affected side. Lie patient down to affected side maintaining 45^0 head rotation and wait for one minute.
- Swing head and body rapidly to the opposite side with head still turned in the same direction. In the new position, the nose is 45 degrees below horizontal. Wait for 2 minutes.
- Raise patient to a sitting position.

Self- treatment of PC_BPPV on the right :

Brandt Daroff positional exercises:

Brandit and Daroff proposed mechanical self treatmnent for PC_BPPV based on Schuknecht's hypothesis of cupulolithiasis in the form of exercises that the patient should perform for 15 minutes 3 times daily.These exercises consist of a rapid sequence of lateral head and body tilts starting from sitting position the patient rapidly moves to the challengimg position

- ,Lying on the affected side (Nose 45^0 up) and remains in this position for atleast 30 seconds or until the vertigo subsides.

- He then sits up for 30 seconds, assumes the opposite head lateral and nose up position for 30 seconds before sitting up.
- Sitting up.

Modified Epley's procedure(FIG10) :

Start by sitting on a bed and turn your head 45 degrees to the rt. Place a pillow behind you so that on lying back it will be under your shoulders. Lie back quickly with shoulders on the pillow, neck extended and head resting on the bed. In this position the affected ear is underneath. Wait for 30 seconds. Turn your head 90 degrees to the left without raising it and wait again for 30 seconds. Turn your body and head another 90 degrees to the left and wait for another 30 seconds. Sit up on the left side. This manoeuvre should be performed three times per day. Repeat this daily until you are free from positional vertigo for 24 hours.

HORIZONTAL CANAL BENIGN PAROXYSMAL POSTITIONAL VERTIGO :

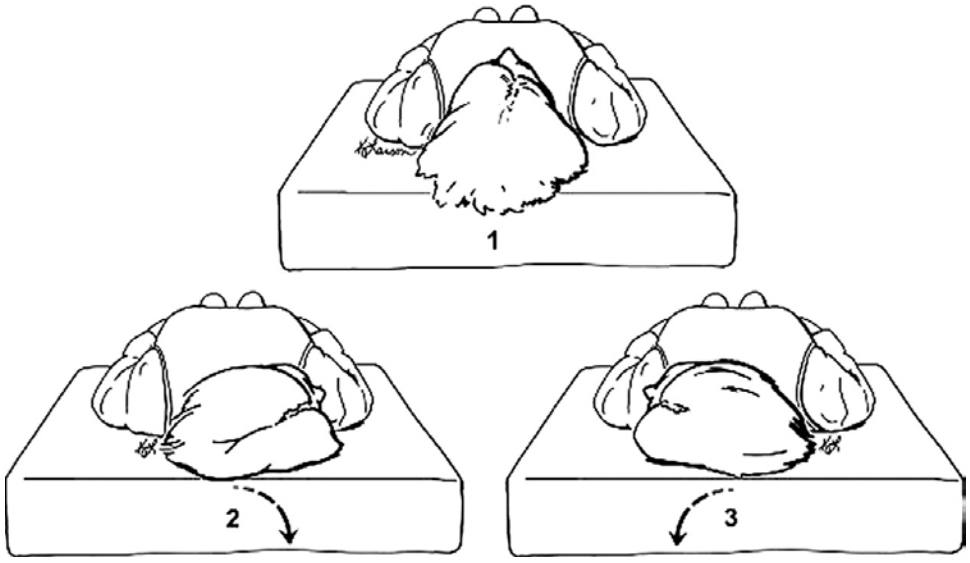
Involvement of the horizontal canal accounts for 10-20% of all patients presenting with benign paroxysmal positional vertigo. Two variants have been described namely the more common ***canalolithiasis*** and the rarer ***cupulolithiasis*** of the horizontal canal. The horizontal canal variants are important to recognize

since they have features that have formerly been attributed to positional vertigo of central origin.

In canalithiasis of the horizontal canal (HSCC-BPPV) attacks of vertigo are provoked by turning the head to either side in the supine or reclined position whereas sitting up or lying down produces only minor symptoms. Similarly, horizontal head movements in the upright position are usually well tolerated,. Symptomatic episodes during which attacks of vertigo are provokable tend to be shorter than with the involvement of the posterior canal, lasting from days to a few weeks. Episodes of (HSCC-BPPV) often alternate with other variants of BPPV.

The supine roll test

. (1) This is a diagnostic positional testing in this patient lies on his back and the head is in the starting neutral nose-up position. The patient's head is turned rapidly to the right side (2) to examine for characteristic nystagmus. Then the head is returned to the face-up position (1), allowing all nystagmus to subside, and then turned rapidly to the left side (3) to examine once again for nystagmus.



Often, the typical nystagmus is elicited also by conventional Dix-Hallpike testing, Eye movements can be easily observed without Frenzel's glasses. The supine lateral head turn provokes transient horizontal nystagmus beating towards the ground regardless of whether the head is turned to the right or left. The typical nystagmus starts with no or minimal latency, beats purely horizontally with respect to the head, lasts up to one minute changes direction when the head is turned to the opposite side and shows no or minimal fatigue with repetitive provocative manoeuvres. When the provoking position is maintained, a less intense but longer lasting nystagmus with reversed direction may appear. Several nystagmus features help to identify the affected side.

1. Nystagmus is always stronger with the head turned to the side of the affected horizontal canal.

2. On this side nystagmus reversal is more pronounced.

3. The change from sitting to supine may provoke a transient horizontal nystagmus to the healthy side, while bending forward usually induces nystagmus to the affected side.

Pathophysiology

Like PC-BPPV the (HSCC-BPPV) variant is caused by aggregated otoconia which have entered a semicircular canal. A head turn to the affected ear while supine induces movement of the otolith and endolymph flow towards the cupula which is located in the anterior portion of the canal. The resulting excitation of the horizontal canal hair cells results in transient horizontal nystagmus beating to the affected side, to the undermost ear. The direction of nystagmus reverses with a head turn to the other side as the clot is shifted in the opposite direction. This type of horizontal nystagmus which beats to the ground in either head lateral position is called geotropic nystagmus. Spontaneous remission after a few days is common in (HSCC-BPPV) because particles may leave the horizontal canal easily during natural head movements. In contrast, the posterior canal is predisposed to become an otolith trap owing to its anatomical position i.e., lower most.

Lateral Canal BPPV Site of Origin and Mechanism

DIRECTION OF NYSTAGMUS		
Intensity of Nystagmus	Ageotropic	Geotropic
Stronger on left side	Right cupulolithiasis	Left canalithiasis
Stronger on right side	Left cupulolithiasis	Right Canalithiasis

In some patients with lateral SCC BPPV, the otoconia are adherent to the cupula (cupulolithiasis) and both the vertigo and nystagmus persist following a Dix-Hallpike manoeuvre. In these cases, the nystagmus is ageotropic-that is, it beats towards the uppermost ear. This clinical picture can suggest a central, rather than peripheral vestibulopathy and may thereby cause diagnostic confusion. Occasionally,it can be difficult to be certain which side is affected. The ageotropic nystagmus is usually more prominent when the culprit ear is uppermost. In addition, the nystagmus is minimal when the head is turned slightly towards the affected side- the so-called ***null head position.***

Manuoeuvres for HSCC:

Prolonged position manoeuvre developed by vanucchi:

He advised patients with HC –BPPV to lie down on the healthy side for 12 hours

to allow the otolithic debris to gravitate to the vestibule by maintaining the affected HSSC uppermost.

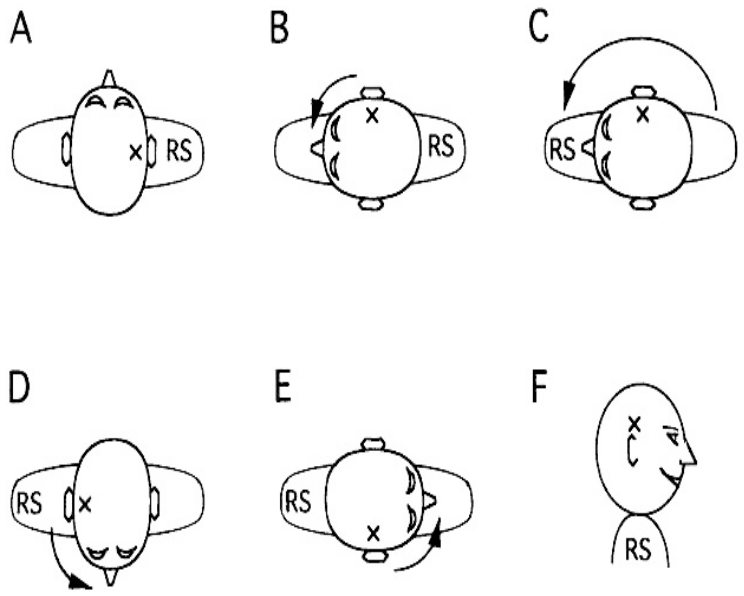
Presence of obesity and cervical spondylosis did not permit maintainance of the position for the time required.

Barrel roll manoeuvre described by Epley :

This involves rolling the patient 360 degrees , supine to supine , keeping the lateral semicircular canal in the earth-vertical plane. The patient is rolled away from affected ear in 90 degree increments until a full roll is completed. This believed to move the particle out of the involved canal in to the Utricle.

Log roll manoeuvre (270^0 '***Barbecue***' or '***Lempert's*** manoeuvre):

The supine patient is rotated 270 degrees in rapid steps of 90 degrees in the plane of the horizontal canal towards the healthy side. The time interval between each step is 30 seconds or until nystagmus has subsided. This positional manoeuvre can be used for self treatment at home. From left to right. Star in the supine nose up position. Turn head 90 degrees to to the left and rotate body by 180 degrees. Turn head to nose down position. Turn head 90 degrees further to the left. Finally, sit patient up to his/her left side.



360^0 yaw rotation manoeuvre:

described by Baloh .360^0 yaw rotation performed in 90^0 steps at 30 second intervels.it should be undertaken until nystagmus disappears.

Gufoni' s manoeuvre:

(1) Patient seated on the examination couch with both the legs hanging out from the same side, arms held close to the body, and hands resting on the knees

(2)Patient is then made to lie down on the uninvolved lateral side with a quick lateral movement and maintained in this position for 2 minutes until the end of evoked geotropic nystagmus

(3)Quick 45% rotation of the head towards the floor, position being maintained for 2 minutes.(slow return back to them starting position

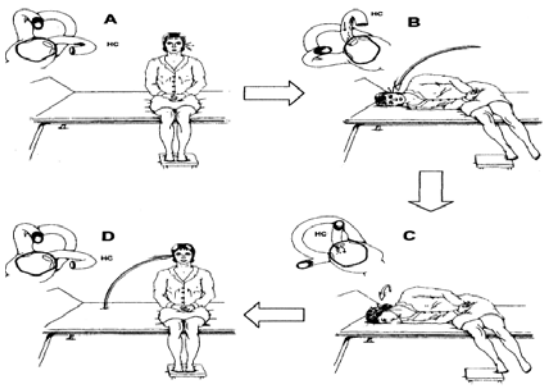
Liberatory manoeuvres;

De la Meilleure manouvre:

From the supine position head is lifted by 30^{0} , turned to the affected side and maintained in that position for 5 minutes. Head is then turned as quickly as possible 180^{0} to the other side and maintained there for 5 minutes. After the manoeuvre, the patient is asked to avoid head shaking and not to lie down for next 48 hours. Contrindications for this manoeuvre are cervical spondylosis, vertebrobasilar insufficiency or neck pain during the manoeuvre

Modified semont's or Appiani's or ***Asperlla*** or C***asani's*** manoeuvre:

In this the patients first sits on the side of the treatment table . He is then quickly brought to lying position on his unaffected and remains in this position for 1 minute after the end of nystagmus. The head is then turned 45^{0} downwards and position maintained for 2 minutes. The patient then slowly returns to the sitting position.



Complications

HSCCcanalolithiasismay convert to cupulolithiasis

Cupulolithiasis to canal plugging -could be managed by vigorous head shake or gentle head percussion

ANTERIOR SEMICIRCULAR CANAL

Anterior semicircular canal BPPV (AC-BPPV) is exceedingly rare . Anatomically, the anterior canal is protected from otoconia debris due to its superior position. If it is involved it is probably due to canalolithiasis as a result of otoconia reflexing in to the canal at the common crus during posterior treatments. Cupulolithiasis of the anterior canal is anatomically infeasible and we have not seen it in our practices. During the Dix-Hallpike test, the anterior canal of the upper most ear is also stimulated . if it is the involved canal, the resulting nystagmus (AC-BPPN) is torsional , with its vertical component beating downward. This is because the anterior canal, when excited , innervates the ipsilateral superior rectus(causing the ipsilateral eye to elevate) and the contralateral inferior oblique(causing the contralateral eye to extort) and the resulting nystagmus is ageotrophic(towards the anterior canal). Currently there is no specific manoeuvre for the anterior canal . However theoretically it can be treated by placing the head more inferiorly while applying mastoid oscillation to move the otoconia towards the vestibule or by performing a reverse Semont's maneuver to move otoconia out of the anterior

canal in to the vestibule. Anterior canal BPPV can sometimes be better elucidated by making the patient lie supine with the head extended in the primary position i.e. straight back or 'Rose' position. Torsional nystagmus with a major downbeat component is seen. The side is indicated by the torsional element beating towards the affected side

Herdman and Tusa succesfuly treated two patients with AC-BPPV with canalith repositioning manoeuvre (right AC-BPPV treated with left CRP and viceversa).Brandit observed that Brandit Daroff exercise were effective and superior to single step manoeuvres for patients treated for a AC-BPPV.

a) Reverse Epley's manoeuvre(Honrubia1999):

1. Sit the patient on examination coach with the head rotated 30 degrees towards the unaffected side

2. Take him backwards to a head hanging position by keeping the head turned 30 degrees to the side and wait for 30 seconds

3. Sit him up again

b)*RAHKO's manoeuvre*: The patient lies on the healthy side, the head is tilted downwards 45 degree, then horizontally, upwards 45 degree for 30 s each, and finally the patient sits up and stays there well supported for at least 3 min.

c) Prolonged forced position procedure (PFPP,L.crevits 2004)

Subjective versus objective BPPV

A certain subset of patients may not demonstrate the typical nystagmus during the Dix–Hallpike manoeuvre, but they may still experience the classic vertigo during positioning. This has been termed “subjective” BPPV, and several studies have found repositioning manoeuvres to be highly

effective in this group of patients. Haynes and colleagues,31 Tirelli and colleagues 32 and Weider and colleagues33 found that patients with subjective BPPV who were treated with various repositioning manoeuvres had response rates of 76%–93% overall. Proposed theories to explain the lack of nystagmus in patients with BPPV during the Dix Hallpike manoeuvre include the following: subtle nystagmus missed by the observer, fatigued nystagmus from repeat testing before the manoeuvre and a less noxious form of BPPV that elicits vertigo but with an inadequate neural signal to stimulate the vestibulo-ocular pathway

Differential Diagnosis:

Meniere’s disease

Inner ear concussion

Alcohol intoxication

Labyrinthitis

Vascular loop syndrome

Posterior Fossa lesions : acoustic neuroma , meningioma

Central origion : stroke , Multiple Sclersis , cerebellar degeneration

PITFALLS

There is no specific manoeuvre to diagnose anterior semicircular canal BPPV.

It is very difficult to differentiate posterior semicircular canal canalithiasis and cupulolithiasis. There will be confusion if the patient is having multiple canal BPPV. In case of bilateral BPPV it is very difficult to identify if it is converted one of disease per se.

PROPOSED THEORIES TO EXPLAIN THE LACK OF NYSTAGMUS

During Dix-Hallpike manoeuvre in BPPV patients

1.Subtle nystagmus missed by the observer

2.Fatigued nystagmus from repeated testing before the manoeuvre

3. A less noxious form of BPPV that elicits vertigo but has an inadequate neural signal to stimulate vestibulo ocular pathway

Factors influencing outcome of repositioning manoeuvre- controversial

-application of mastoid oscillator

-prior use of vestibular sedative

- use of frenzel's glass

-Following up of post manoeuvre instructions

-Presence of other etiological factors like vestibular neuronitis ,meniere's disease etc

SURGICAL TECHNIQUES

Less than 1% of patients with BPPV will require surgical treatment as a last resort.

1.*Posterior semicircular occlusion*

2.Singular neurectomy

3. Partitioning of labyrinth

Indications for surgery

Recurrent or chronic symptoms that are sufficiently debilitating to warrant an invasive procedure. Patient should demonstrate conservative therapy failure, including repeated failures of particle repositioning maneuvere and persistence of symptoms for atleast one year.

ContraIndication for surgery

Acute or subacute episodes of otitis media

COMPLICATIONS

1.Hearing loss

2.Post operative disequlibrium

3.tinnitus

MATERIALS AND METHODS

STUDY PLACE **:** Upgraded Institute of Otorhinolaryngology
Madras Medical College
Government General Hospital, Chennai - 600 003.

STUDY DESIGN **:** Prospective

STUDY PERIOD **:** JULY 2008 TO SEPTEMBER 2009.

INCLUSION CRITERIA :

1. Age group 14yrs to 60 yrs
2. Clinical evidence suggestive of positional vertigo.-BPPV
3. Patients willing for the study

EXCLUSION CRITERIA:

1. Age more than 60 yrs and age less than 14yrs
2. Patients on labyrinthine sedative
3. Bilateral BPPV
4. Multicanal pathology
5. Patients with acute illness like fever, hypoglycaemia
6. Patients with neck problem like cervical spondylosis
7. Patients with other causes of vertigo
8. Patients not willing for the study

Patients who presented with giddiness were selected with a detailed questionnaire regarding history and were meticulously assessed about the onset, duration and position at which patient develops vertigo and they were subjected to otological examination, tuning fork test, pure tone audiometry.

All patients were subjected to vestibular function tests like Romberg's test, tandem walking, Fukuda's stepping test and caloric test to confirm peripheral vertigo. They were advised to stop any labyrinthine sedative medicines at least 3 days prior to examination. Those who are taking it for long time were given with placebos.`Adjuvant supports like mastoid oscillation frenzel's glass, Dizzy fix (is a dynamic visual device which is used to complete the manoeuvre in proper way)were not utilized.

Dix Hallpike positional test, head roll tests were done as per the clinical guidance. Eye movements were directly visualized without the aid of defocusing lenses. If the direction of nystagmus reversed during the manoeuvre, the procedure was halted and repeated. The treatment efficiency was evaluated by obtaining subjective relief of the patient which was graded (I to III). This was noticed by the patient in his dairy for two weeks when it is relieved completely

The diagnosis of posterior semicircular canal BPPV was confirmed by observation of upbeating torsional nystagmus when the head was moved in the

plane of the posterior semicircular canal. Anterior canal BPPV was identified by a downbeating torsional nystagmus and horizontal canal BPPV by a direction changing horizontal nystagmus after brisk horizontal head movements.

The presence of latency and a crescendo-decrescendo pattern of the nystagmus were mandatory for the diagnosis. The details of nystagmus were critically assessed and the side and exact canal of involvement were identified and according to canal involvement, therapeutic manoeuvres like Epley's, Semont's procedure were done as an office procedure and outcome (vertigo)was graded based on severity of symptoms and signs. Forced position manoeuvre was advised for lateral canal and during follow up also, outcome (vertigo) were graded after the manoeuvre at 4^{th} day,10^{th} day,1 month and 6 months.

Position test was done and patients were classified in to 3 types.

Those patient who are not resolved the manoeuvre like Semont's or Epley's repeated. Patient was followed for a second 2 weeks.

For the patients those who are I grade iii and type iii were advised to undergo investigations like special audiological tests, CT and MRI to rule out other causes.

INSTRUCTIONS FOR PATIENTS AFTER CANAL REPOSITIONING TREATMENT

1. Wait for 10 minutes after the manoeuvre is performed before going home. This is to avoid “quick spins” or brief bursts of vertigo as debris repositions itself immediately after the manoeuvre. If possible, have someone drive you home.

2. Sleep slightly elevated for the next 3 days. This means sleep on two pillows so that you are not completely horizontal. During the day, try to keep your head vertical. You must not go to the hairdresser or dentist. Avoid exercise which requires excessive head movement. When men shave under their chins, they should bend their bodies forward in order to keep their head vertical. If eye-drops are required, try to put them in without tilting the head back. Shampoo your hair only under the shower.

3. For at least 3 days, avoid provoking head positions that might bring on an episode of vertigo.

_ Use two pillows while sleeping

_ Avoid sleeping on the “bad” side

_ Don’t extend your head far up or flex it far down

_ Be careful to avoid head-extended positions in which you are lying on your back, especially with your head turned towards the bad side. This means be cautious at the beauty saloon, dentist’s office, and if having minor surgery. Ask them to keep

you as upright as possible. If appropriate, exercises for low back pain should be discontinued for three days. Avoid sit-ups and “crawl stroke” swimming for three days. (Breast stroke is OK)

_Avoid far head-forward positions such as might occur in certain exercises like touching the toes.

4. At 3 days after treatment, put yourself in the position that usually makes you dizzy. Position yourself cautiously and under conditions in which you can’t fall or hurt yourself. Let your physical therapist know how you did.

PROFORMA

"EFFICACY OF PARTICLE REPOSITIONING MANOEUVRE IN BENIGN PAROXYSMAL POSITIONAL VERTIGO".

GENERAL INFORMATION:

Name		**Hospital no**	
Age /sex		**Serial no**	
Occupation		**Date**	
Address			
Contact no		Others	

GIDDINESS

ONSET	DURATION	EPISODES	DURATION OF EACH EPISODE	AGGRAVATING FACTORS	RELIEVING FACTORS	REMARKS

GRADING OF GIDDINESS

Grade 0	No giddiness
Grade 1	Walks without support
Grade 2	Walks with support
Grade 3	Unable to get up from bed

H/O RTA/head collision

H/o LRI/URI

H/O Ear surgery

H/O Previous drug intake

Labyrinthine sedative

Antihistamine

Sedative

H/o Neck pain

Associated Problem: HT/DM/CAHD/Neck pain

GENERAL EXAMINATION

Comfortable

Vital signs

Gait

EXAMINATION OF EAR

NOSE

THROAT

NECK-APPEARANCE

-MOVEMENT

FISTULA TEST

VESTIBULAR TESTS:

Romberg's test

Tandem Romberg

Tandem walking

Fukuda's stepping test

CALORIC TEST:

GRADING OF DIX- HALLPIKE TEST

Grade 0	Without nystagmus and giddiness
Grade 1	With giddiness without nystagmus
Grade 2	With nystagmus and giddiness

INVESTIGATIONS:

PTA:

X RAYS:

CT/MRI:

BLOOD INVESTIGATIONS:

DOPPLER:

OTHERS:

PROVISIONAL DIAGNOSIS: BPPV

Rt PSCC	**Rt ASCC**	**RT LSCC**	**LT PSCC**	**LT ASCC**	**LT LSCC**

PARTICLE REPOSITIONING MANOEUVRE DETAILS:

Type of procedure performed:

Date of first procedure:

Post procedure subjective symptomatic benefit: grade 1 2 3

Dates of second procedure:

Date of third procedure:

FOLLOW UP

FOLLOW UP	4^{TH} day	10^{th} day	1month	6month
Symptoms [Grade]	1 2 3	1 2 3	1 2 3	1 2 3
Dix hall pike [type]	1 2 3	1 2 3	1 2 3	1 2 3

COMPLICATIONS ENCOUNTERED: [IF ANY]

RESULTS AND ANALYSIS:

Demographic details:

Fifty patients were included in the study, males were 19 (38%), females 31 (62%).(table2&chart2) female to male ratio is 1.64:1.Their age ranged between 14 and 60 years (37 years) (table1&chart1). All the patients had giddiness and were found to have a positive Dix-Hallpike/supine roll test confirming the clinical diagnosis of BPPV. In five patients (10%), nausea and vomiting were found to be associated.

Symptoms:

Two (4%) patients had history of trivial head injury. None of the patients had history of preceding viral URTI, or any associated visual, cervical or neurological complaints. Four patients (8%) were diabetic.

The patients were asked to score the negative impact of vertigo on the quality of their life and their daily activity. Pre-PRM, 11 patients (48%) gave a score of 10 (very badly affected), while post- PRM 21 patients (88%) scored 0 (No effect at all on their life). Twenty-two patients (91.7%) admitted a significant improvement post-therapy (Table&chart7).12(24%) were presented with very severe form of vertigo and responded very well for PRM and got immediate relief and walked out

TABLES

TABLE 1:AGE DISTRIBUTION

AGE	14-20	21-30	31-40	41-50	51-60
NUMBER	6	5	15	13	11
PERCENTAGE	O.12	O.1	O.3	O.26	O.22

TABLE 2: SEX DISTRIBUTION

MALE	FEMALE
19	31

TABLE 3: SIDE DISTRIBUTION

RIGHT	LEFT
33	17

CHARTS

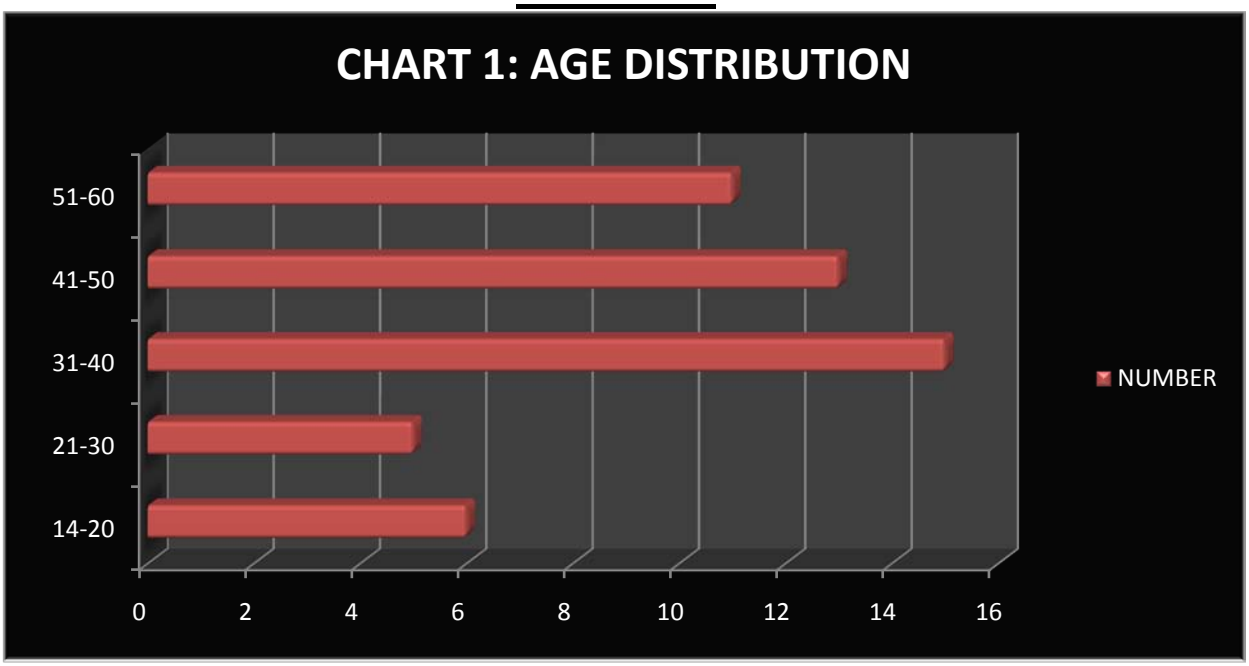


CHART 2: SEX DISTRIBUTION

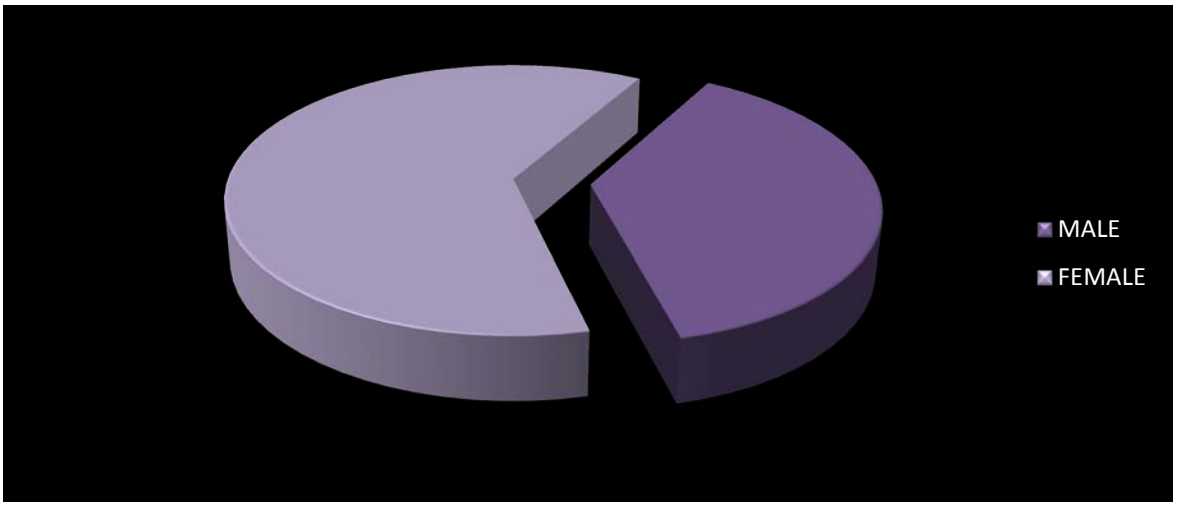


CHART 3 : SIDE DISTRIBUTION

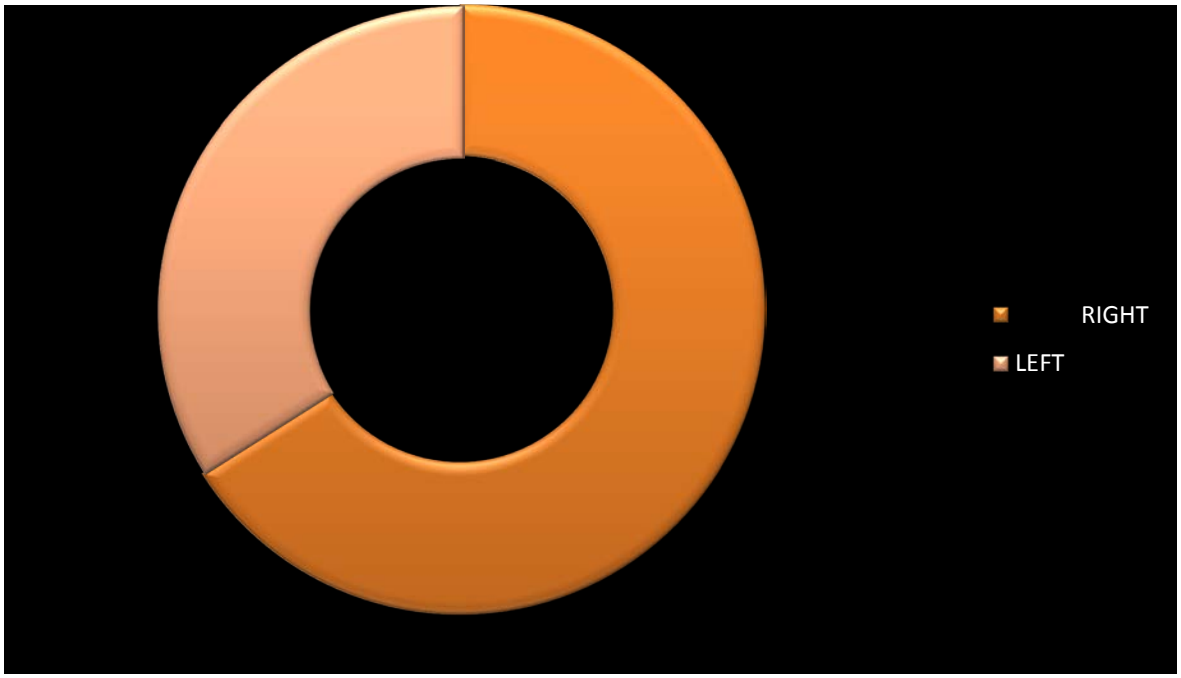


CHART 4 : DURATION DISTRIBUTION

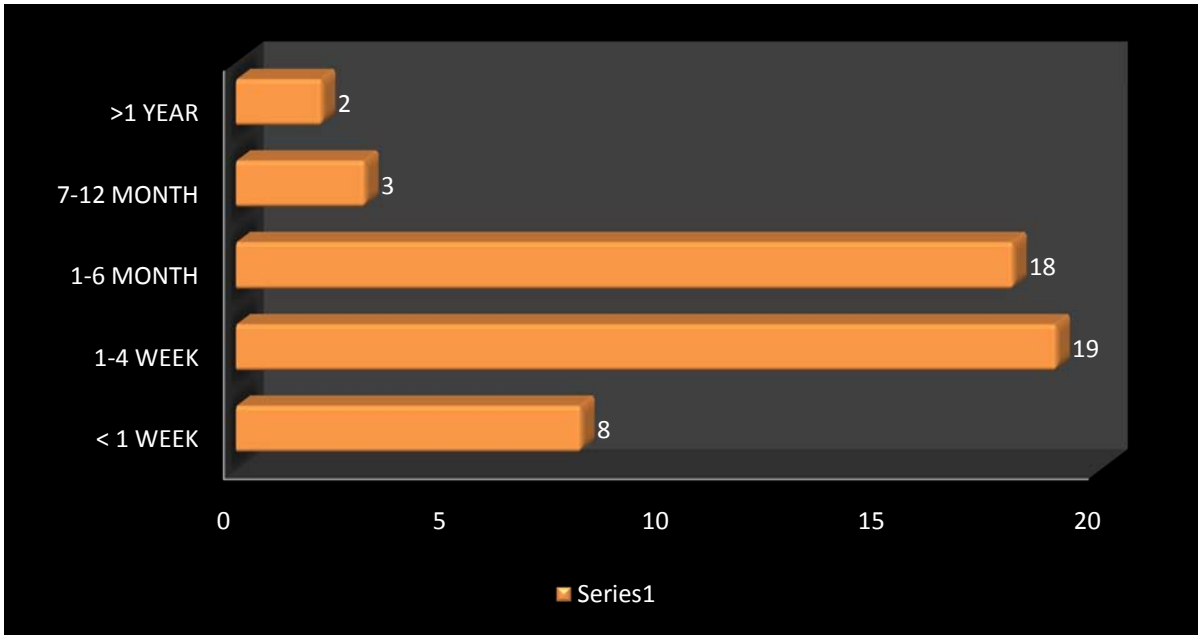


TABLE 4: DURATION DISTRIBUTION

DURATION	NO.OF PATIENTS
< 1 WEEK	8
1-4 WEEK	19
1-6 MONTH	18
7-12 MONTH	3
>1 YEAR	2

TABLE 5:CANAL DISTRIBUTION

POTERIOR SCC	LATERAL SCC	ANTERIOR SCC
45	4	1

TABLE 6: PARTICLE REPOSITIONING MANOUEVERS

EPLEY	FPP	SEMONT	REVERSED EPLEY
43	4	2	1

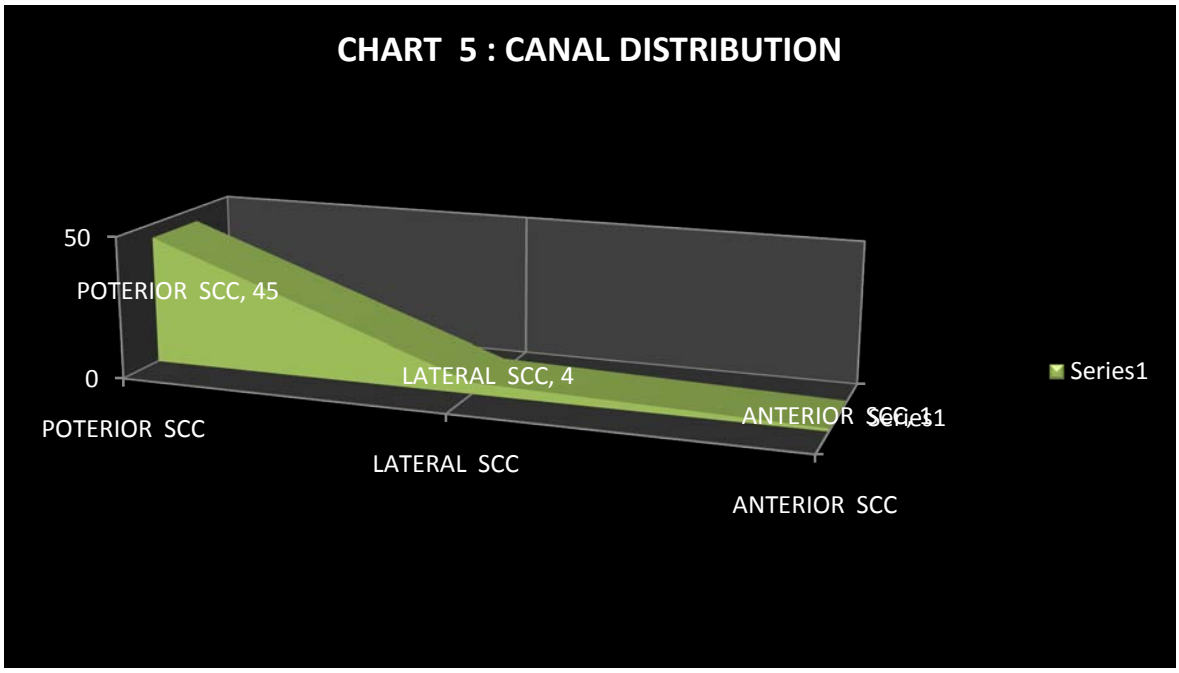


CHART 6: PARTICLE REPOSITIONING MANOUEVERES

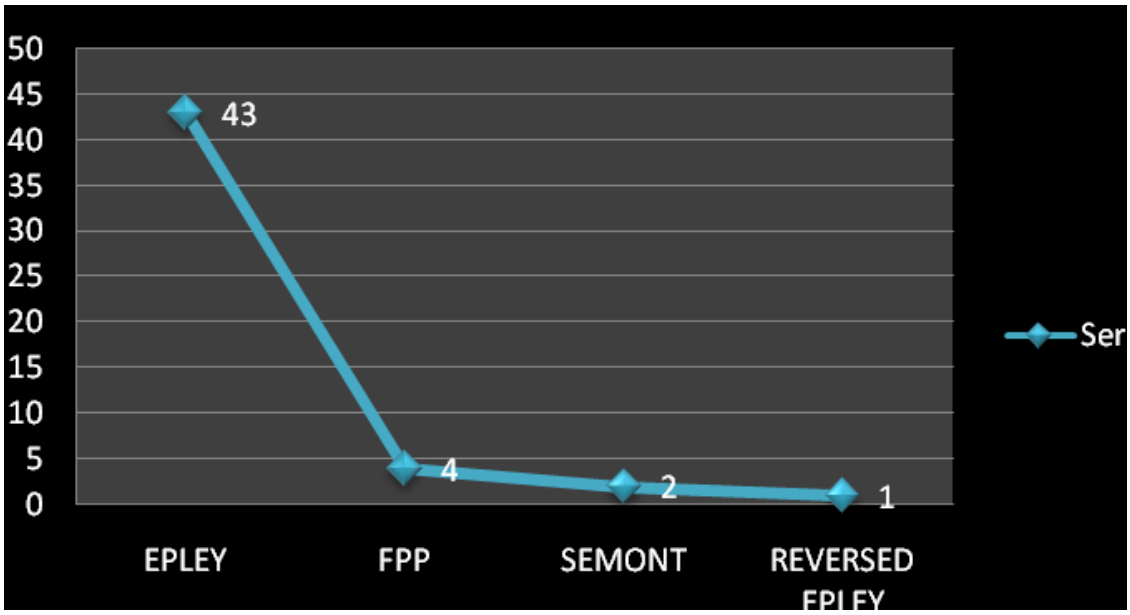


CHART 7 :SUBJECTIVE GRADING

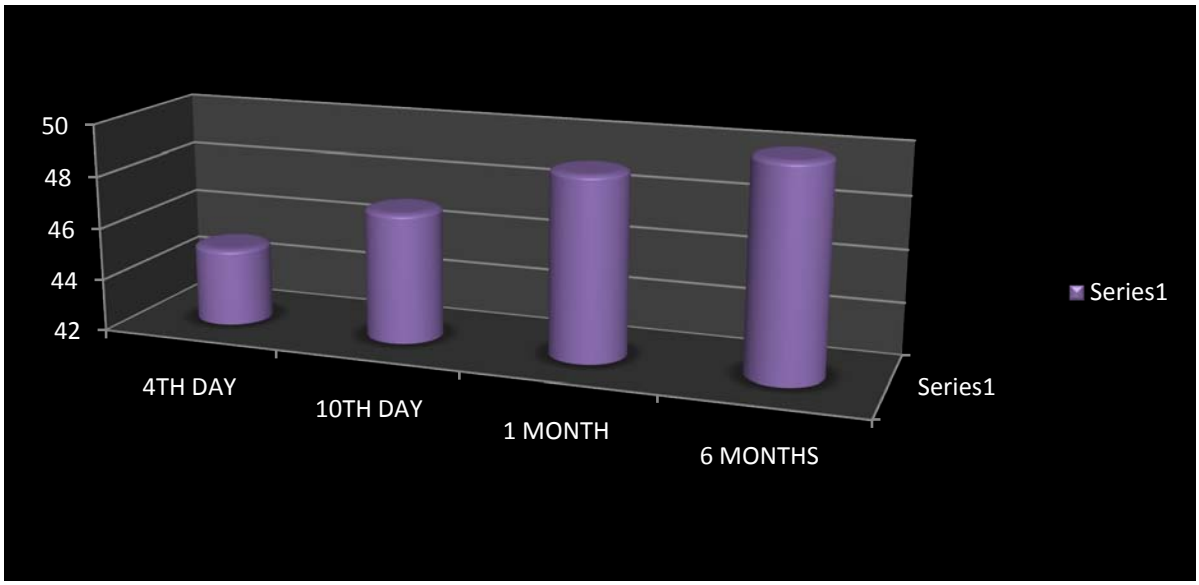
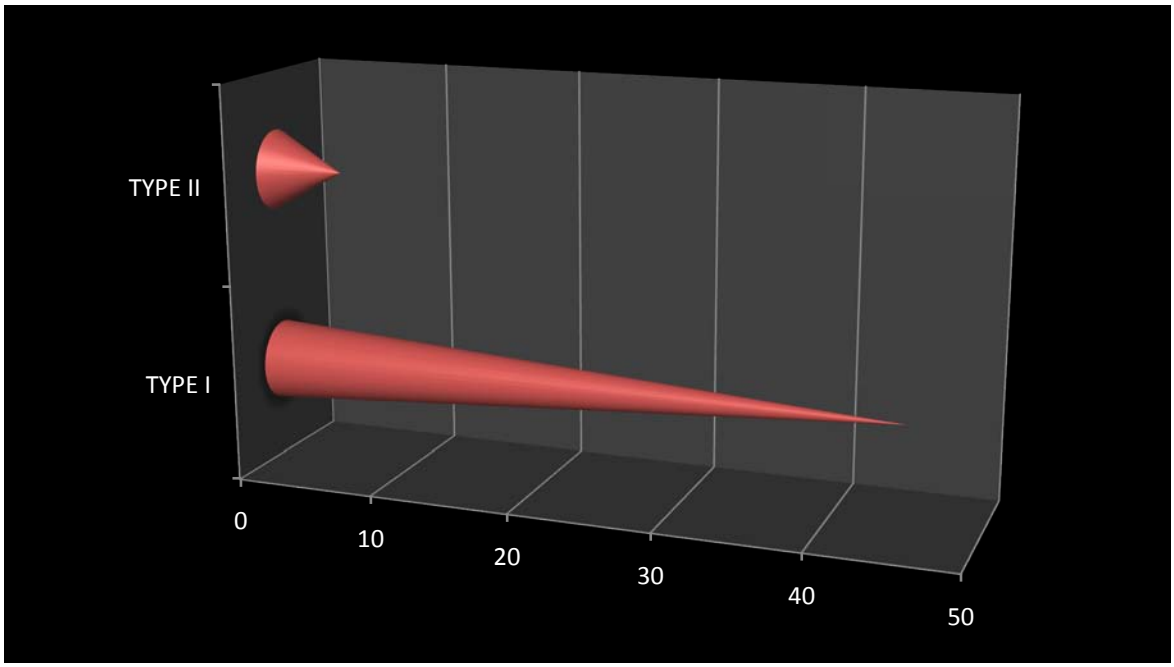


CHART 8: OBJECTIVE RELIEF GRADING



comfortably from outpatient department.

The follow up period was for a minimum of six months. All the patients claimed a strict compliance with post-PRM manoeuvre's instructions after the procedure. When interviewed at the time of conducting the study, the vast majority of the patients claimed a dramatic improvement reflected in complete cessation of vertigo in 45 patients (90%). In the remaining 5 patients, 4 patients showed improvement at the end of 1 week and one patient claimed no relief at all. Even though literature claims up to 30% of recurrence within one year in our study no patient had recurrence within this six months follow up.

DISCUSSION

In this study, it was able to clearly demonstrate the effectiveness of various particle repositioning manoeuvre (PRM) in the treatment of BPPV, with success rate approaching 90%. Other single-session of Epley's manoeuvre publications, their success is usually ranging from 78 - 90% .An overlooked important aspect of the BPPV is its impact on the quality of life, daily activity, and the dramatic improvement achieved post-therapy, was addressed and stressed in this study.

Subjective assessment of BPPV patients treated with Epley's manoeuvre in different studies[14]:

S.NO	Author	Year	No of patients	Follow up 7 days	15 days	1 month
1.	Asawavichianinda et al.	1998	35	88.6%	88.6%	94.2%
2.	Harvey et al.	2000	94	60.6%	78%	-
3.	Dal	2000	68	72%	88.2%	91.1%
4.	Khatri et al.	2003	34	53%	82.3%	85.2%
5.	Present Study	2009	50	90%	95%	-

Objective assessment of BPPV patients treated with Epley's manoeuvre in different studies[14,26]:

S.No.	Author	Year	No. of	Type I response	Type II response	Type III response
1.	Epley	1992	30	90%	10%	-
2.	Nunez and Cass	1996	151	91.3%	7.9%	0.8%
3.	Jose et al.	2000	51	83%	9%	-
4.	Verma	2001	30	90%	10%	-
5.	Khatri et al.	2003	34	88.2%	8.8%	2.94%
6.	Present Study	2009	50	90%	10%	2%

Sex distribution in this study concurs with the literature review, which suggests that BPPV is more common in females (62%), which is comparable to 62.5% in Epley's et al.

The recurrence rate of BPPV after particle repositioning manoeuvre was nil within the following year and 4 patients required a second session. Some authors modified the original procedure aiming to achieve extra success in the outcome by shaking or tapping the temporal region of the affected ear during the procedure, or using vibrators placed over the mastoid bone. Others, however, modified the procedure by increasing the degree of head rotation, or the duration of each head position. Furthermore, some studies claimed better outcome by repeating the procedure within a week. In this study, we followed the original non-modified

Epley's Manoeuvre, and still our outcome was within if not better than many of the modified techniques. Daniel et al. reported a study on the management of Bilateral Benign Positional Paroxysmal Vertigo (BiBPPV) in which they described a typical characteristics of BiBPPV and confirmed that they can be managed successfully with Epley's manoeuvre performed on the most symptomatic side, followed by repeated treatment as needed. Epley classified the result after treatment with the manoeuvre into: I. Resolution of vertigo, II. Presence of nonpositional vertigo,III. Partial resolution and IV. Same or worse.

On the other hand, Herdman et al classified the result into: 1. No symptoms, negative Dix-Hallpike manoeuvre, 2. Improvement with abnormal manoeuvre, 3. No change.

SUMMARY

Patients presenting with giddiness should be carefully screened by detailed questionnaire regarding onset, duration, predisposing factors and associated other problems. History of treatment with vestibular sedative drugs should be elicited , as it interferes with diagnosis and treatment. Then we should do ENT examination, tuning fork tests, pure tone audiogram and vestibular function tests . Then positional tests like Dix-hallpike and supine roll test done, the nystagmus watched carefully, semicircular canal involvement diagnosed. Posterior semicircular canal most commonly involved and it is prevalent in middle aged female with right sided predominance.

After diagnosing posterior canal BPPV Epley's manoeuvre used primarily, at second visit Epley's or Semont's manoeuvre done. For horizontal canal BPPV, diagnostic manoeuvre is supine roll test and therapeutic manoeuvre forced prolonged position manoeuvre is used. In anterior canal BPPV we had difficulty in diagnosis and treatment.

After doing appropriate therapeutic manoeuvre patients were asked to wait for half an hour and given with post manoeuvre instruction.

Patients were followed up on 4th day, 10th day ,one month and sixth month. Even though literature showed 30% recurrence rate at one year in our study we didn't get any recurrence within the follow up of six months period.

Correctly diagnosed and appropriately executed particle repositioning manoeuvre will give maximum benefit to the patients.

CONCLUSION

Particle Repositioning Manoeuvres are found to very effective procedures in the management of isolated BPPV affecting a single semicircular canal.

Since history and clinical examination are the only tools in the diagnosis of BPPV, a proper evaluation will help in careful localization of the semicircular canal affected.

- Most commonly occurs in middle aged females. Female male ratio is 1.63 :1.
- Posterior semicircular canal involvement is most common.
- Right side is predominant to left.
- It presents with episodic vertigo, occurs in particular position and lasting for seconds to minutes
- Elicitation of history and positional test is adequate to diagnose
- Particle repositioning manoeuvre is effective way to treat and give immediate relief to the patients even though it is a self limiting disease.

Appropriate particle repositioning manoeuvres when correctly preformed will give the maximum expected benefit to the patients.

S.NO	NAME	AGE	SEX	HOSPITAL NO.	DURATION OF GIDDINESS	POSITION	DIAGNOSIS (BPPV)	TREATMENT MANOUVERE	OUTCOME SUBJECTIVE (GRADE)	OUTCOME OBJECTIVE (TYPE)
1	Chellamuthu	60	M	314/08	15D	L	LtPSCC	L EPLEYS	I	I
2	Saradha	42	F	318/08	6D	R	RtPSCC	R EPLEYS	I	I
3	Nataraj	38	M	324/08	1W	R	RtPSCC	R EPLEYS	I	I
4	Meenachi	37	F	376/08	1W	R	RtPSCC	R EPLEYS	I	I
5	Parthasarathi	48	M	388/08	2W	R	RtPSCC	R EPLEYS	I	II
6	Sudhakar	56	M	391/08	20D	R	RtPSCC	R EPLEYS	I	I
7	Nagarajan	53	M	523/08	3M	L	LtPSCC	L EPLEYS	I	I
8	Elanjian	60	F	551/08	1Y	L	LtPSCC	L EPLEYS	I	I
9	Mallaikannan	41	M	630/08	6M	L	LtPSCC	L EPLEYS	I	I
10	Palaniyamal	40	F	651/08	3W	R	LtACC	R EPLEYS	III	III
11	Saravanan	14	m	663/08	2M	L	LtPSCC	L EPLEYS	I	I
12	Saridha	35	F	680/08	6D	R	RtLSCC	RFPP	I	I
13	Kamarunisha	45	F	686/08	3y	L	LtPSCC	L EPLEYS	I	I
14	Kasthuri	59	F	714/08	7M	L	LtPSCC	L EPLEYS	I	I
15	Sundhari	40	F	717/08	3M	L	LtPSCC	L EPLEYS	I	II
16	Jayapaulraj	35	M	736/08	1D	R	RtPSCC	R EPLEYS	I	I
17	Savithri	41	F	737/08	2M	L	LtPSCC	L EPLEYS	I	I
18	Vedhammal	45	F	758/08	1W	R	RtPSCC	R EPLEYS	II	II
19	Suja	30	F	760/08	15D	R	RtPSCC	L EPLEYS	I	I
20	Vasantha	45	F	762/08	2D	R	RtPSCC	R EPLEYS	I	I
21	Jayanthi	35	F	777/08	4D	R	RtPSCC	R EPLEYS	I	I
22	Parasakthi	50	F	795/08	2M	L	LtLSCC	LFPP	I	II
23	Arumugam	55	M	110/09	3M	R	RtLSCC	L FPP	I	I
24	Amudha	48	F	244/09	2M	L	LtPSCC	L EPLEYS	I	I
25	Prabakar	22	M	356/09	2Y	r	RtPSCC	L EPLEYS	I	I
26	Subramani	60	M	362/09	10D	r	RtPSCC	L EPLEYS	I	I
27	Vijaya	55	F	402/09	1Y	R	RtPSCC	R EPLEYS	I	I
28	Lakshmi	21	F	496/09	10D	R	RtPSCC	R EPLEYS	I	I
29	Rajeswari	38	F	507/09	4D	R	RtPSCC	R EPLEYS	I	I
30	Thenmozhi	32	F	589/09	6M	R	RtPSCC	R EPLEYS	III	III
31	Sarala	40	F	618/09	3W	R	RtPSCC	R EPLEYS	I	I
32	Nagalakshmi	18	F	621/09	5M	L	LtPSCC	L EPLEYS	I	II
33	Puspha	45	F	704/09	1M	R	RtPSCC	R EPLEYS	I	I
34	Kumari	29	F	728/09	1M	R	RtPSCC	R EPLEYS	I	I

35	Keerthana	19	F	744/09	3M	L	LtPSCC	L EPLEYS	I	I
36	Anandhari	16	M	806/09	6M	R	RtPSCC	R EPLEYS	I	I
37	Sridevi	36	F	848/09	1M	R	RtPSCC	R EPLEYS	I	I
38	Saravanan	15	M	854/09	1M	L	LtPSCC	L EPLEYS	II	I
39	Rajasekar	53	M	918/09	1M	R	RtPSCC	R EPLEYS	I	I
40	Ganagavalli	32	F	965/09	10D	R	RtPSCC	R EPLEYS	I	I
41	Sankarakumar	40	M	985/09	3D	L	LtPSCC	L EPLEYS	I	I
42	Murugayee	53	F	986/09	2W	R	RtLSCC	RFPP	I	I
43	MohanaRangan	25	M	995/09	1W	R	RtPSCC	R EPLEYS	I	I
44	Shanthi	58	F	1012/09	2W	R	RtPSCC	R EPLEYS	I	I
45	Shek bhasha	60	M	1019/09	10D	L	LtPSCC	L EPLEYS	I	I
46	Dulasi	40	F	1039/09	3D	R	RtPSCC	R EPLEYS	I	II
47	Irusammal	17	F	1063/09	1W	R	RtPSCC	R EPLEYS	I	I
48	Nagaraj	46	M	1068/09	5M	R	RtPSCC	R EPLEYS	I	I
49	Gopal	47	M	1086/09	3W	R	RtPSCC	R EPLEYS	I	I
50	Greeda	40	F	1080/09	1W	L	LtPSCC	L EPLEYS	II	I

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X)ABBREVIATIONS

BPPV- benign paroxysmal positional vertigo

PRM-particle repositioning procedure

CRP- canalith repositioning procedure

SCC- semicircularcanals

PSCC-posterior semicircularcanals

HSCC-horizontal semicircularcanals

ASCC-anterior semicircularcanals

VOR-vestibular ocular reflex

VSR-vestibulospinal reflex

XI) CONSENT FORM

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

"சாதாரண திடீர் நிலை சார்ந்த தலைசுற்றலில் துகள் மறுநிலைமபடுத்துதல் முறைகளின் பயன்பாடு"

மேம்படுத்தப்பட்ட காது, மூக்கு, தொண்டை உயர்நிலைத்துறை : சென்னை மருத்துவக் கலலூரி சென்னை - 600 003.

பங்கு பெறுபவரின் பெயர் :
பங்குபெறுபவரின் எண் :

பங்கு பெறுவர் இதனை (✓) குறிக்கவும்.

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்மந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக்கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்க்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்கிறேன். ☐

இந்த ஆய்வில் எனக்கு இரத்தம், சிறுநீர், எக்ஸ்ரே, ஸ்கேன் பரிசோதனை செய்துகொள்ள நான் முழு மனதுடன் சம்மதிக்கிறேன். ☐

பங்கேற்பவரின் கையொப்பம் ..... இடம் ..... தேதி

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி

ஆய்வாளரின் பெயர் .....

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XIII) ETHICAL COMMITTEE CERTIFICATE